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ABSTRACT

Nutrition is well-recognized as a necessary component of educational programs for physicians. This is to be valued in that of all factors affecting health in the United States, none is more important than nutrition. This can be argued from various perspectives, including health promotion, disease prevention, and therapeutic management. In all cases, serious consideration of nutrition related issues in the practice is seen to be one means to achieve cost-effective medical care. These modules were designed to provide more practical knowledge to health care providers, and in particular primary care physicians. This module is designed to present nutrition-related consequences of chronic consumption of alcohol. The effects of alcohol on nutrient metabolism as well as nutritional intervention when liver metabolism is altered secondary to alcohol ingestion and liver damage are discussed. Since the patient who continues to drink poses special nutritional problems, suggested counseling techniques are presented. Included are learning goals and objectives, self-checks of achievement with regard to goals, and references for the physician and for the physician to give to the patient. (CW)

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14 Dietary Management for Alcoholic Patients

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Nutrition in Primary Care

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14 Dietary Management for Alcoholic Patients

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Introduction

Acute or chronic excessive consumption of alcohol is a major problem in the United States today. Alcoholism has been defined as a complex series of disordered and often destructive behaviors, characterized by repeated drinking of alcoholic beverages to the extent that consumption interferes with the patient's health, interpersonal relationships, and economic functioning. Chafetz chose to classify alcohol consumption into 3 states:[2]

1. Alcohol abuse — occasional heavy drinking caused by a problem occurring at the time but which is not typical behavior.

2. Problem drinking — heavy drinking which may affect work, home, and social situations; this occurs when the drinker feels a need to cope or to relieve pressures.

3. Alcoholism — frequent, consistent, and intense overuse of alcohol that consumes the individual's life.

As many as 11 million people are considered problem drinkers. The extent of alcoholism is significant, especially when one considers that the alcoholic is not just the skid-row variety. In fact, fewer than 5% are in the class in which the individual's economic, physical, social, and emotional resources are drained such that feelings of helplessness and nihilism prevail.[2]

The occurrence of excessive drinking in adolescents is growing. An estimated 19% of persons 14 to 17 years old are considered problem drinkers, generally "binge" drinking on weekends. Teenagers are influenced by parental role models, peer pressure, environmental pressures such as inadequate financial resources, and by personal factors such as a sense of alienation and deviative behavior that may predispose the adolescent to drink. Effects of alcohol abuse in adolescents include school and job-related problems and psychological problems often associated with drug abuse.[2,12]

Although there are fewer women than men drinking excessively, the estimates of problem drinking among women are probably conservative, since the drinking can occur in the privacy of the home. Like adolescents, women who drink are more likely to abuse drugs than are non-drinkers.[12]

The physician can notice changes in the life of a woman patient that might make her a higher risk for heavy alcohol consumption — major changes in her family life, or a "trapped" feeling at home. When a woman works outside the home, she may also feel a need to conform to the male model where daytime drinking is common.[2] Because females absorb alcohol more rapidly premenstrually and because the alcoholic

female may drink to decrease premenstrual tension, heavy alcohol use at this time may increase chances of alcohol dependency.[12] Drinking during pregnancy conclusively has been found to increase the incidence of miscarriages and abortions. It also affects the development of the fetus and child — fetal alcohol syndrome — in terms of low birth weight, facial abnormalities, joint and limb anomalies, and mental retardation.[2]

Surveys have shown that problem drinking in the elderly occurs in 10% of men and 2% of women. Contributing factors that are "red flags" to you include loneliness, loss of spouse, poor health, and feelings of uselessness with lack of occupation.[2]

The effects of chronic excessive ingestion of alcohol on the individual include general physical and mental deterioration. The fatty liver, hepatitis, and cirrhosis of alcoholism and subsequent malnutrition and metabolic changes are well documented. Cirrhosis is the sixth leading cause of death in the US. Drinking poses problems with the vocational, marital, physiological, and psychological aspects of one's living. Some of the problems are:

- Chronic illness or disability.
- Acute health problems related to specific drinking bouts.
- Injuries, death, and property losses caused by accidents and crimes.
- Failure of the drinker to fulfill family and job roles.
- Mental problems such as depression and anxiety.

Innocent members of society are affected by alcohol in terms of homicides, child and spouse abuse, and violence.[2]

The health care dollars required to treat alcohol-related problems is presently estimated to be $13 billion annually and growing. An estimated $15 to $20 billion is lost in business and industry with employee-related drinking problems, including accidents and time missed from work. Another $10 billion is lost in motor vehicular accidents, in violent crimes, and in social responses.[2,18]

Alcoholism is not without nutritional side effects. The substitution of alcohol for a balanced diet contributes to malnutrition. Unless alcoholism is diagnosed early, the drinker may not receive appropriate nutritional and medical treatment.

Goals

The purpose of this module is to present nutrition-related consequences of chronic consumption of alcohol. The effects of alcohol on nutrient metabolism as well as nutritional intervention when liver metabolism is altered secondary to alcohol-ingestion and liver damage are discussed. Since the patient who continues to drink poses special nutritional problems, suggested counseling techniques are presented. As a result of this unit of study, you should be able to:

1. *Describe consequent nutritional effects of altered nutrient metabolism due to the physiological changes seen with chronic ingestion of alcohol;*

2. *Screen for malnutrition associated with chronic ingestion of alcohol;*

3. *Propose dietary modifications necessary with specific metabolic aberrations due to liver damage;*

4. *Apply the principles of optimum nutrition to the patient who continues drinking as well as the patient who abstains; and*

5. *Counsel the patient who drinks, using the principles of normal nutrition and making the counseling plan realistic for the patient.*

A care study format is used to accomplish these objectives.

Care Study

Mrs. A.J., a 48-year-old housewife, has come to you for a checkup. Her height is 5 feet 4 inches, and her current weight is 105 pounds. During your routine examination, you note that her weight has dropped since her last visit one year ago. Her weight loss represents a loss of 15 pounds or about 12% of her ideal body weight. "Ideal body weight" equals 120 pounds (100 pounds for the first 5 feet plus 5 pounds for each additional inch over 5 feet). She appears pale and somewhat depressed. Because of her weight loss, you take a quick diet history.

The patient reports she has been eating one meal per day for some time. Her husband has been working late and eats dinner with business clients. She fixes breakfast for her family, which includes juice, eggs, bacon, and toast. She straightens up the house early, then has little to do the rest of the day. She "feels low," so she often has a drink to "pick her up" around 11:00 a.m. She usually does not feel like fixing lunch and will often have another drink while watching television in the afternoon. She frequently does not bother with dinner for herself although she does prepare the evening meal for her two children. When she does eat dinner, it is usually an egg and toast. She states that when the kids are home from school, they fight "continuously" and this "drives her nuts." She is tired of housework and is depressed about being "stuck" at home. A friend was concerned about her depression and convinced her to take Norpramin. She also takes a multivitamin with iron supplement since she feels she is not eating right and knows she looks pale.

You ordered the following lab work and found these results:

Hgb	10.8 grams/deciliter
Hct	32%
RBC	3.6 million/cubic millimeter
MCV	100 cubic microns
MCH	30.7 micromicrograms
MCHC	33.1%
Iron	50 micrograms/deciliter
Iron binding capacity	250 micrograms/deciliter
Creatinine	1.3 milligrams/deciliter
Glucose	101 milligrams/deciliter
BUN	21 milligrams/deciliter
Uric acid	7.1 milligrams/deciliter
Cholesterol	242 milligrams/deciliter
Total protein	8.1 grams/deciliter
Albumin	3.1 grams/deciliter
Alkaline phosphatase	4.0 units
LDH	360 units
SGOT	118 units
SGPT	80 units
Na	135 milliequivalents/liter
K	4.6 milliequivalents/liter
Cl	98 milliequivalents/liter

If Mrs. A.J. had not admitted her drinking, what would be some "red flags" that would cause you to suspect she drank? "Red flags" may include:

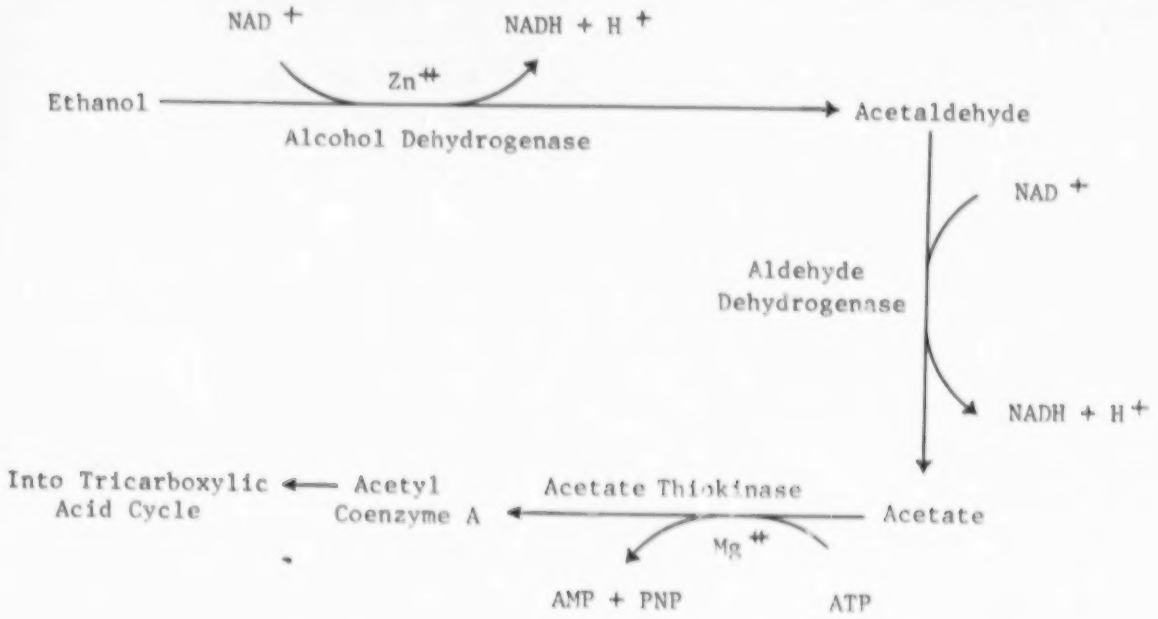
- Weight loss.
- Depression about her home situation.
- Comments about her feelings concerning her children's behavior and her being "stuck at home."
- Comments concerning her lack of good eating habits.

Further questions you might ask include the following:

- What is her typical daily intake of food and beverages?
- Why does she skip meals? Does she substitute any food or drink for a regular meal?
- How does she respond to the children's fighting and her husband's absences?
- How does she cope with her depression? Does she sleep well at night?
- Do any family members or friends abuse alcohol or any other drug?

If your patient works, you will want to inquire about her happiness with the job, stress on the job, and financial affairs.

Once the patient admits drinking, it becomes easier to elicit information regarding quantities of alcohol consumed. Under what circumstances and for what reasons does the patient drink? So-



cially? To escape problems? To relax? To cope? Remember, you must be non-judgmental in your questioning, or physician-patient rapport will suffer.

Physiological Effects of Alcohol

Alcohol has a wide variety of physiological effects on the digestive and absorptive capacities of the gastrointestinal tract.

Do you recall the metabolism of alcohol? It is rapidly absorbed from the stomach and the small intestine and is transported to the liver by the portal vein. Alcohol is 90% oxidized in the liver by the following reaction:[3,10]

Many of the important sequelae of ethanol metabolism are due to NADH (reduced nicotinamide-adenine dinucleotide) accumulation.

What factors could increase Mrs. A.J.'s concentrations of blood alcohol? Factors include:

- Fast drinking.
- High concentration of alcohol per drink.
- No food present in the stomach at the time of alcohol intake.
- Low tolerance to alcohol.
- Alcohol in carbonated beverages such as sparkling champagne is more quickly absorbed than alcohol in non-carbonated drinks.

Alcohol is a known gastric stimulant, causing release of secretin and histamine which, in turn, stimulate release of hydrochloric acid from the stomach's parietal cells. Thus, one may see irritation and, eventually, ulceration of the gastric mucosa which has been observed as a high incidence of gastritis, hemorrhage, and duodenal ulcers in alcoholic patients.

The increased stimulation of digestive secretions can also lead to pancreatitis eventually diminishing the availability of pancreatic digestive

enzymes, fluid, and electrolytes. Mrs. A.J. did not report any of these problems, but you might ask other patients if they have gastritis, diarrhea, or steatorrhea. Steatorrhea is occasionally encountered in the alcoholic secondary to abnormal bile salt metabolism due to liver disease. Chronic consumption of alcohol has been found to decrease daily excretion of bile salts and bile acid pools. Alcohol causes changes in intestinal motility and in the ultrastructure and absorptive functions of the small intestine.

There are many nutritional effects related to the physiological changes seen in alcoholism. These effects include changes in appetite, digestion, absorption, and utilization of nutrients.

Malnutrition in the alcoholic is caused primarily by inadequate intake of essential nutrients and energy. Malnutrition can also result when malabsorption of nutrients occurs due to alcohol's toxic effect on the liver, intestine, and pancreas.

Alcohol decreases appetite. The nausea of binge drinking is secondary to decreases in gastric motility and emptying and contributes to anorexia and diminished food intake. The effects of alcohol on the esophagus are generally related to the increase in gastric acid secretion. Relaxation of the lower esophageal sphincter may occur secondary to high gastric acid secretion leading to heartburn and reflux. Irritation of the esophageal mucosa also occurs with retching. The gastric mucosal irritation caused by alcohol can further depress appetite.

The pancreatitis associated with prolonged alcohol consumption can decrease digestion. Lack of pancreatic lipase causes defective micellar incorporation of fat and fat-soluble compounds. Alcohol also decreases availability of bile salt. Steatorrhea and weight loss can result. Diarrhea reported in binge drinking is caused by the increase in small intestine motility and fluid absorption. The overall effects of alcohol on nutrients are decreased absorption of amino acids, thiamin, vitamin B_{12}, folic acid, and calcium. Water, electrolyte, glucose, and fat absorption are also impaired.

Liver Damage and Nutritional Sequelae

In the early stages of drinking, fatty liver and altered blood lipids and glucose may be seen. Protein status may also be affected.

The changes in liver structure in the drinker vary with the severity and duration of drinking. Two causes of fatty liver development in the alcoholic have been suggested. The first cause is that influx of fatty acids from adipose tissue to the liver is greater than fatty acid conversion to triglyceride and subsequent export from the liver. Causes of increased influx in the alcoholic could be caloric intake less than expenditure (alcoholic starvation) or a decreased ratio of insulin to glucagon. The second cause of fatty liver infiltration in the alcoholic may be delayed clearance of hepatic fat secondary to alcohol consumption. Would you expect to see some hepatic fat infiltration in Mrs. A.J.? Yes, probably. A fatty liver is reversible with cessation of alcohol intake and return to a nutritious diet.

If there is increased triglyceride production secondary to alcohol intake, would you expect blood lipid levels to increase or decrease? Hyperlipidemia is commonly seen in the chronic drinker and is due to an increase in production of very low density lipoprotein. There is additionally a change in lipoprotein metabolism. Interestingly, alcohol has been shown to increase the concentration of the high density lipoproteins (HDL) which may exert a protective effect against vascular changes seen in coronary heart disease. Therefore, alcohol *may* be protective — but only to a point. Yano, et al. found a strongly negative correlation between consumption of alcohol and incidence of coronary artery disease among moderate drinkers (11 to 60 milliliters alcohol per day or the equivalent of 1 beer or 1 ounce whiskey daily). This correlation decreased in nondrinkers, heavy drinkers, and ex-drinkers.[19] In severe liver disease, hypolipidemia is common; lipoprotein synthesis is impaired (and serum triglycerides decrease) at blood alcohol concentrations above 250 to 300 milligrams/deciliter.[10] Hypoglycemia is common in the drinker, occurring with depletion of glycogen stores and inhibition of gluconeogenesis with chronic

consumption of alcohol. Alcoholic patients have also been shown to have elevated insulin levels and abnormal responses to glucagon. On the other hand, hyperglycemia is also seen in some alcoholics secondary to impaired glucose tolerance.

Another early change in liver derangement with prolonged consumption of alcohol is decreased hepatic protein synthesis. Albumin, because it constitutes 50% of total plasma proteins, is highly reflective of the status of total protein. Mrs. A.J. had a slightly depressed serum albumin level, although total protein was within normal limits. Changes in albumin levels are usually not seen in "binge" drinking because of the slow turnover time of albumin. Other proteins not adequately synthesized because of alcohol's toxic effects include transferrin, fibrinogen, prothrombin, and other transport proteins causing susceptibility to infection and depressed blood vitamin and mineral levels. Chronic intake of alcohol along with malnutrition also decreases testosterone levels causing typical feminization in the male drinker.

The interrelationships between dietary intake and the development of alcoholic hepatitis and cirrhosis are not clear.

Malnutrition has previously been proposed as the predominant factor in the pathogenesis of alcoholic liver disease. This proposition has been based upon the indirect evidence of frequent observations of poor nutritional status of hospitalized alcoholics with liver disease as well as the inadequate dietary intake these patients report. Much evidence has, however, accumulated in the past two decades to support the view that alcohol is a direct toxin to the liver and as such is the major cause of liver disease in the alcoholic. In clinical studies, alcohol exerts morphologically and biochemically toxic effects regardless of dietary variations in fat, protein, vitamins, and lipotropes (choline and methionine).

When alcoholic hepatitis is present, large doses of alcohol, seen in "binge" drinking, may override the protective effects of an optimal diet. Animal studies have shown this "loading" effect may play a role in the development of the typical lesions of hepatitis and cirrhosis.[15]

While there is no solid evidence that cirrhosis is caused by dietary deficiency, the incidence of cirrhosis correlates with the duration and amount of alcohol consumed. In a number of studies, cirrhosis was seen in 25% to 50% of persons drinking more than 160 grams of alcohol per day for 15 to 25 years. No relationship to diet composition was seen although it is realized that alcoholics typically consume a diet low in protein and food energy. In a study of 304 chronic alcoholics, the cirrhotics consumed 15% less protein and food energy than non-cirrhotics prior to onset of disease. The fact that so many drinkers do not develop cirrhosis indicates that factors other than poor diet contribute to cirrhosis.

There are a number of laboratory tests that reflect changes in liver structure although they generally do not have nutritional implications. Serum transaminases, dehydrogenases, and phosphatases are examples of such tests. Both SGOT (serum glutamic oxalacetic transaminase) and SGPT (serum glutamic pyruvic transaminase) increase early in hepatitis or cirrhosis. Mrs. A.J. had elevations of both enzymes; this indicates some liver cell damage. Lactic dehydrogenase, LDH, was also elevated in Mrs. A.J.'s blood and is a nonspecific indicator of cellular damage. Alkaline phosphatase, another isoenzyme in the liver, was also elevated, a typical finding in liver disease, especially cirrhosis.

The ascites and edema in cirrhosis are due to a low blood protein level secondary to dietary deficiency or inadequate protein synthesis. With depressed blood albumin levels, blood colloid oncotic pressure decreases, and fluid accumulates in the peritoneal cavity and extravascular spaces. The patient's dietary protein needs are therefore elevated.

With progression of liver disease, a variety of metabolic effects may occur including changes in hematologic, infectious, cardiovascular, and neurologic functions.

Hematologic effects of prolonged consumption of alcohol are varied. Alcohol has a direct toxic effect on bone marrow production of red blood cells, hemoglobin synthesis, and on iron, folate, and vitamin B_{12} metabolism. Lack of hemoglobin, accumulation of iron in the bone marrow,

and folate deficiency are factors in the development of sideroblastic anemia. Since alcohol decreases absorption of vitamin B_6 and prevents its activation to pyridoxal phosphate, heme-synthesis is impaired. The resulting anemia is microcytic and hypochromic. *Megaloblastic anemia* is seen when folic acid and vitamin B_{12} are deficient and red cells cannot mature. An indirect effect of alcohol is reduced folic acid absorption. This leads to a *macrocytic, megaloblastic anemia. Iron-deficiency anemia* (microcytic, hypochromic) is generally secondary to blood loss, gastritis, bleeding esophageal varices, and peptic ulcer disease. Alcoholics are usually not iron-deficient. Instead, alcoholics may be especially prone to hepatic injury secondary to excess iron storage. Because alcohol increases gastric acidity, iron absorption increases. In addition, alcoholic beverages may contain considerable amounts of iron.[6]

What were Mrs. A.J.'s values for hemoglobin, hematocrit, RBC count, MCV, MCH, and MCHC? Her low hematocrit with high MCV suggests a reduced number of RBC's and macrocytosis. Low hemoglobin with normal MCH suggests reduced heme-synthesis but normal amounts of hemoglobin per red blood cell. Normal MCHC indicates a normal ratio of hemoglobin to red blood cell size. Had you checked red blood cell folate and vitamin B_{12}, you would have found her folate level was depressed and vitamin B_{12} was normal. Both folic acid and vitamin B_{12} levels should be checked when MCV is greater than 95 cubic microns. Her anemia is of the megaloblastic type with high MCV and normal MCH and MCHC.

The patient's serum iron level was low; why is she not iron-deficient? A protein that is not produced when protein status is low or when the liver is damaged is transferrin, the transport protein for iron. Because Mrs. A.J.'s transferrin level is reduced, her iron binding capacity is also low, as would be expected. The low transferrin level would account for her depressed serum iron levels. A normal serum ferritin would confirm this; this test is helpful in diagnosing anemia in chronic diseases such as alcoholism. Infectious diseases such as pneumonia, tuberculosis, and subbacterial endocarditis have been related to consumption of alcohol. It is felt that in alcoholics there is a reduction in the components of cell-mediated immunity, resulting in increased susceptibility to infection and lowered immune response. There is frequently a nonspecific increase in the gamma fraction in chronic liver disease. IgA and IgG are frequently increased in patients with alcoholic liver disease.[9]

Cardiovascular changes in the alcoholic occur because of alcohol's direct toxic effect on the myocardium; specifically, alcohol and acetaldehyde both interfere with cardiac protein synthesis. Elevated blood pressure is also seen with prolonged drinking in men and women taking 3 or more drinks a day. ECG changes best demonstrate the pathological lesions of heart tissue.[1,17] ECG changes following drinking include increased S-T segment depression with exercise. Common abnormalities include S-T segment and T-wave changes, as well as sinus tachycardia.

The neurological changes produced by alcohol may be direct or may be a combined result of many factors. The Wernicke-Korsakoff syndrome is associated with changes in thiamin-dependent enzymes caused by low thiamin intake, decreased thiamin stores, and decreased thiamin activation to thiamin pyrophosphate in the damaged liver. Other thiamin-related problems include alcoholic polyneuropathy and cerebellar degeneration. Patients with ocular manifestations of Wernicke's encephalopathy have delayed response to thiamin administration if cirrhosis is present. Thiamin deficiency may be only one cause of encephalopathy. Table 14–1 lists other causes, presumed mechanisms, and therapy.[5,8]

The "culprit" in the central nervous system (CNS) changes of encephalopathy is unclear; certain amines and abnormal amino acid patterns due to competition of aromatic and branched-chain amino acids for entry across the blood-brain barrier have been implicated. Ammonia itself may be a "marker" that indicates abnormalities rather than actually being the toxic substance.[4]

Chronic consumption of alcohol with insufficient caloric intake may cause muscle breakdown or *myopathy.* Labile proteins may be used to meet the body's demand for glucose and energy. Ketosis and dehydration may contribute to the development of acute renal failure. In addition, excess lactate production associated with excess of NADH + decreases renal clearance of uric acid, causing hyperuricemia and symptoms of gout.[3] Mrs. A.J. had a slight increase in serum uric acid.

Table 14–1 Precipitating Causes of Encephalopathy with Nutritional Implications

Causes	Mechanism and Treatment
Azotemia and Hyperammonemia	Increased enterohepatic circulation of urea nitrogen with increased ammonia; treat with nonabsorbable antibiotics (Neomycin) or Lactulose to decrease gut flora synthesis and blood reabsorption of ammonia.
Metabolic alkalosis	Increased diffusion of ammonia across blood-brain barrier; give supplemental potassium if alkalosis is caused by hypokalemia.
Excess dietary protein	Substrate for ammonia and production of other nitrogenous toxins; reduce dietary protein, especially aromatic and sulfur-containing amino acids that have high ammonia-generating potential.
Constipation	Increased production and absorption of ammonia with increased contact time of bacteria and nitrogenous substances. Straining upon defecation may increase portal pressure and chances of varices. Treat with stool softeners and laxatives.

Nutritional Management in Liver Disease

Generally, nutritional therapy for alcoholic patients should include a diet which is low enough in protein to prevent encephalopathy yet supplies enough protein and calories to prevent tissue catabolism. Repletion of vitamin and mineral status, if necessary, is essential.

You have a number of tools available to assess the nutritional status of the alcoholic patient. Use of the diet history is invaluable:

- Has the patient had a recent change in appetite, weight, or bowel movements?
- How many meals per day are eaten?
- What foods constitute a meal?
- Is the patient on a limited income? How much money is spent on food and alcohol?
- Who prepares the patient's meals?
- Does the patient drink more than 4 ounces of liquor per day?

Comparison of the patient's typical intake to the Basic Four Food Groups guidelines can tell you if the intake is optimal. What about Mrs. A.J.'s typical intake? You know that the Basic Four suggests intake of the following for the non-pregnant adult female:

Meat — two 2-ounce servings per day
Milk — 2 servings per day (8 ounces milk or equivalent equals one serving)
Fruits and Vegetables — 4 servings per day
Breads and Cereals — 4 servings per day (1 slice or equivalent equals one serving)

Mrs. A.J.'s intake as reported includes:

Meat — 1 serving per day
Milk — none
Fruits and Vegetables — 1 serving per day
Breads and Cereals — 2 to 3 servings per day
Alcohol — at least 3 ounces per day

You should have assessed that her intake is low in energy, protein, and essentially all vitamins and minerals. The vitamin supplement with iron which she takes does help meet her Recommended Dietary Allowances (RDA) for vitamins and iron but probably not for folate or for minerals other than iron. Without adequate protein and energy, utilization of other nutrients will be impaired.

What are Mrs. A.J.'s nutrient needs? *Energy* needs must be met by replacing alcohol with nutritious, high calorie foods. The RDA for energy for the non-pregnant adult female is approximately 30 to 40 kilocalories per kilogram ideal body weight. Our patient is below her ideal body weight of 55 kilograms; therefore, she should increase her intake of energy to approximately 40 to 50 kilocalories per kilogram per day. This increase is also appropriate for tissue regeneration and protein-sparing. How many kilocalories was Mrs. A.J. receiving from 3 ounces alcohol per day? At about 7 kilocalories per gram of alcohol, she was consuming approximately 650 kilocalories per day in alcohol, which is approximately 30% of her RDA for energy. Table 14–2 includes the alcohol and caloric content of several beverages.

The alcoholic's need for protein depends on the patient's liver function. Serum protein levels may be depressed because of inadequate protein intake. Do you recall the two major reasons why protein levels may be depressed in the drinker? The patient may have inadequate intake or decreased hepatic synthesis of various proteins. When hapatitis is present, high intake of protein (1.0 to 1.5 grams protein per kilogram body weight) is recommended to restore synthesis of the labile tissue proteins which are highly responsive to changes in nitrogen and caloric intake. If cirrhosis is present, the patient's ability to metabolize protein may be compromised. In patients with uncomplicated cirrhosis, protein intake should be reduced to 0.7 grams protein per kilogram body weight per day.

With complicated cirrhosis such as elevated blood ammonia, hepatic encephalopathy, or coma, the patient should be hospitalized and a zero gram protein diet begun. Following a drop in blood ammonia level to normal and return of consciousness or improvement of flapping tremor, intake of protein may be increased, first to 10, then to 20, to 30, and, finally, to 40 grams protein per day or greater. A 10 gram increase every other day should be instituted until tolerance is reached and the liver is unable to titrate additional amounts of ammonia. Titration tolerance will be the maximum amount of dietary protein a patient should be allowed (usually 0.4 to 0.7 grams protein per kilogram body weight). The protein ingested by the patient should be of high biological value (egg, milk, beef, or other meats). The caloric intake must be adequate to spare protein from being used as an energy source.

When beginning oral intake following coma, it is usually best to begin day one with 1 egg or 8 ounces milk (7 to 8 grams protein respectively) plus high carbohydrate-supplemented juices served cold at the bedside for the patient to sip frequently and continuously during the day. On the second or third day a second egg or 8 ounces milk should be added to increase protein by another 8 grams. A half cup cereal along with the milk could also be supplied. Consult with a registered clinical dietitian at this time as, in addition, you must meet the patient's caloric needs through use of special low-protein, high-carbohydrate, low-electrolyte products which are available in hospitals and which patients with a low protein tolerance may need to buy when at home. Special low-protein flours, starches, and pastas may be purchased to augment a low-protein diet. For example, low-protein bread, cookies, cakes, and other baked goods can be made from the following products:

Paygel-P wheat starch and dietetic Paygel-P Baking Mix. Write to:

Henkel Corporation
4620 West 77th Street
Minneapolis, MN 55435
or
Aprotein Pasta Diet Erba S.P.A. of Latina, Italy
Henkel Corporation
4620 West 77th Street
Minneapolis, MN 55435

Table 14–2 Alcohol and Caloric Content of Selected Beverages

Beverage	Serving Size	Alcohol Content (grams)	Kilocalorie Content
Ale, mild	8 ounces	8.9	98
Beer, regular, average	8 ounces	8.9	114
Beer, average, light	8 ounces	9.6	66
Brandy, California	1 brandy glass= 30 cc	10.5	73
Creme de Menthe	1 cordial glass= 20 cc	7.0	67
Daiquri	1 cocktail glass= 90 cc	15.1	122
Eggnog, Christmas	4 ounces	15.0	335
Gin, dry	1 jigger= 45 cc	15.1	105
Highball	8 ounces	24.0	166
Manhattan or Martini	1 cocktail glass= 90 cc	19.0	150
Mint Julep	10 ounces	29.2	212
Rum	1 jigger= 45 cc	15.1	105
Tom Collins	10 ounces	21.5	180
Whiskey, Scotch	1 jigger= 45 cc	15.1	105
Wine, Champagne	1 wine glass= 4 ounces	11.0	84
Wine, Sherry	1 sherry glass= 60 cc	9.0	84

Cellu-Low Protein Mix
Chicago Dietetic Supply House
405 East Shawmut Avenue
LaGrange, IL 60525

Recently, studies have been reported of using infusions of parenteral keto acids and total parenteral nutrition with a low amino acid content on liver-damaged patients. The theoretical advantage of the keto acid approach is that keto analogues combine with amino donor groups to form essential amino acids, provide no nitrogen load, and reduce hyperammonemia. Results include improvement in the ratio of essential to non-essential plasma amino acids, reduction of plasma glutamine levels, and improvement in mental status. Nitrogen balance has not been shown to be appreciably affected. Unfortunately, the therapy is expensive, and the amount of sodium and calcium which are present in the keto acid solution may be problematic for the severely liver-damaged patient with disordered metabolism of fluid and electrolytes.

The recommendation to try total parenteral nutrition (TPN) with liver disease patients has come from three major lines of reasoning:

1. The diminished tolerance to dietary protein;
2. The data suggesting altered amino acid requirements in liver-diseased patients; and
3. The implication of altered amino acid patterns in the pathogenesis of hepatic encephalopathy.

In patients with hepatic encephalopathy, elevated levels of blood methionine and aromatic amino acids and decreased blood branched-chain amino acids have been shown. Such an effect may be explained by the fact that the liver metabolized aromatic amino acids (which are decreased with liver damage) and that muscle is the primary site of branched-chain amino acid metabolism (which is

not decreased during liver disease). Therefore, TPN solutions high in branched-chain amino acids and low in aromatic amino acids have been tried with success in rectifying plasma amino acid imbalances, helping maintain positive nitrogen balance and improving mental status.

Dietary fat may need to be restricted in liver-diseased patients who have decreased ability to digest and absorb fat. Undue dietary restriction of fat, however, makes the diet unpalatable and poses difficulty in consuming adequate kilocalories to spare the limited intake of protein. The ability of the patient to tolerate fat should be tested, and a 30 gram restriction implemented only if needed. A more feasible restriction would be to limit obvious fats including butter, margarine, oils, fried foods, and creamed desserts. Skim milk should be substituted for whole milk. Use of medium-chain triglycerides (MCT) in patients with liver disease is not wise as these fatty acid chain lengths (8 to 12 carbons), although requiring less pancreatic lipase and bile than long-chain fatty acids for absorption, are metabolized by the liver and pose the damaged liver additional metabolic problems.

The alcoholic patient needs a multivitamin supplement with folic acid. Ascites requires rigid control of sodium intake.

It is generally felt that supplementation of all the vitamins and minerals is appropriate in the alcoholic patient since vitamin and mineral depletion is common. A one-a-day multivitamin/multimineral supplement plus folic acid is recommended. Problems associated with folic acid, pyridoxine, thiamin, and vitamin B_{12} have been discussed earlier in this module. In particular, alcohol alters utilization of most of the B vitamins and affects storage of niacin. Conversion of cholecalciferol to 25-hydroxycholecalciferol is delayed in the presence of cirrhosis. This abnormality in vitamin D metabolism may contribute to the decreased calcium absorption and osteoporosis seen in alcoholics with cirrhosis. Decreased liver conversion of carotene to vitamin A and decreased storage of retinol occurs in liver damage and can cause vitamin A deficiency resulting in visual cycle changes. Because the liver is the site of synthesis of clotting factors dependent on vitamin K, hepatocellular damage results in decreased synthesis of these factors, prolonged prothrombin time, and bleeding diathesis. Poor prognosis is associated with failure of vitamin K administration to correct a prolonged prothrombin time.

Mineral deficiencies may be caused by poor intake, maldigestion, and increased excretion. The alcoholic may have increased urinary excretion of zinc and magnesium. Calcium absorption and utilization are impaired with lack of active vitamin D. Serum iron may be low, but you must guard against iron-overload.

Chronic liver disease due to alcoholism may cause disorders of water and electrolyte metabolism. We have already reviewed ascites and edema caused by hypoalbuminemia. Water and sodium retention may also be caused by portal hypertension, altered lymph flow, decreased renal function, and endocrine changes. Rigorous sodium restriction may be required on the order of 250 to 500 milligrams per day depending upon the degree of ascites and hypertension. With less severe fluid retention, sodium may be restricted to 500 to 2,000 milligrams per day. Fluid restriction is necessary if hyponatremia is evident. Daily weights are essential; weight loss due to fluid excretion should not exceed 5 kilograms per week by diet and diuretic therapy. Potassium depletion occurs with vomiting, diarrhea, diuretic therapy, muscle wasting, and altered renal function. Spironolactone treatment for ascites may result in increased potassium reabsorption from the kidney and therefore obviate the need for potassium administration. Hypokalemia must be avoided as it contributes to increased production of renal vein ammonia with worsening of hepatic encephalopathy.

Of course, the ideal outcome of treatment of the alcoholic patient is the cessation of intake of alcohol and institution of a well-balanced diet. Prevention of drinking is problematic since it requires identification and treatment of the causes of drinking. Most likely a reduction in intake of alcohol is all that can be achieved. The patient must be encouraged to plan nutritious meals during those times of the day when he or she is not drinking. The eating of at least one balanced meal per day and the eating of as many nutritious foods as possible between drinking bouts should be encouraged.

Interactions of alcohol and nutrient utilization are well documented in Module 3 on nutrient and drug interactions. It is essential to work with the patient so that he is aware that alcohol may interfere with normal nutritional status and that lack of optimal dietary intake of kilocalories and other nutrients can have serious health consequences.

If appetite or motivation to prepare meals is a problem, the patient should be encouraged to take smaller, more frequent feedings. Use of nutritious beverages such as milk, milkshakes, eggnogs, or commercial supplements may be used as between meal feedings. Increased awareness of when and why binge drinking occurs can help the patient work out an appropriate feeding schedule.

Treatment programs for the alcoholic have been developed to educate and provide a team approach to dealing with the complex problems associated with alcoholism. Some are detoxification programs only. Others go beyond this service and provide counseling, a dimension important not only for the alcoholic but also for the family.[18]

Effective treatment programs must include nutritional counseling in which the patient receives personalized counseling and attention. Including the family or significant others in the program is important as they play an important role in rehabilitation. The nutritional education component of the program should include follow-up efforts.

How would you go about counseling Mrs. A.J.? First of all, you need to help Mrs. A.J. take a look at her daily intake of food. She is substituting alcohol for food. She is bored and concerned about her husband's absences. She appears to have little self-esteem and obviously needs some self-fulfillment. With some probing, you discover that she worked to put her husband through school, and they began their family immediately after he went to work. This meant that she did not attend college which she would like to have done. Perhaps she should consider returning to school now and fulfill her dreams of becoming an attorney. Most state colleges and universities have degree and non-degree programs to meet older persons' career choices.

The cycle of drinking at home must be broken. You suspect that the patient underestimates her drinking, which is probably true. Her husband, you later learn, is more than happy to play a more supportive role to help his wife.

Mrs. A.J. has a long way to go until she is totally rehabilitated. It is doubtful she will go "cold turkey." Based upon her pattern of drinking, you suggest she continue her breakfast intake. While she is cleaning the house in the morning, suggest she begin to prepare lunch and dinner so that it is ready at these times and the house is full of kitchen-aroma. A casserole with meat and vegetables would improve intake from these two food groups. Preparing custard or pudding, which are good sources of protein, might improve the chances of her consuming foods from the milk group. The idea is that she should have foods ready when mealtime arrives. It may take her some time to return to three meals per day, but the attempt should be made. Follow-up nutritional counseling by a clinical dietitian is advisable to provide emotional support for the patient and to help motivate her to gain weight and prevent potential dietary deficiencies.

Summary

Throughout this module, we have stressed the importance of early diagnosis of alcoholism and have suggested screening tools for your use. Careful nutritional assessment of the alcoholic patient depends on the degree of liver damage. For example, if malabsorption is present, replacement of those dietary constituents that have been lost is essential. If weight loss and anorexia occur, small, frequent, yet high-caloric feedings are necessary; if gastritis or ulcer symptoms are complaints, your patient should avoid gastric stimulants and other gastrointestinal irritants. If malnutrition is obvious, then high kilocalorie and supplemental vitamin and mineral intake should be stressed. **Most of all, the patient should stop drinking since even an optimal diet cannot combat the deleterious effects of alcohol.**

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Resources for the Physician

Roe, D.A.: *Alcohol and the Diet*. Box 831, Westport, CT 06880. AVI Publishing Company, 1979, 228 pages. (hardbound, $15.00)

In this book, the author discusses the interrelationships of alcohol intake and nutritional status. She provides practical guidelines for nutritional assessment and rehabilitation of alcoholic patients and offers insights into some behavioral studies.

Some Abbrevations Used in the Nutrition in Primary Care Series

ATP	adenosine triphosphate
c	cup
cc	cubic centimeter
CNS	central nervous system
FDA	Food and Drug Administration
gm	gram
IBW	ideal body weight
IU	International Units
kcal	kilocalorie
kg	kilogram
lb	pound
lg	large
MCV	mean corpuscular volume
MDR	minimum daily requirement
med	medium
mEq	milliequivalent
mg	milligram
MJ	megajoule
ml	milliliter
oz	ounce
RDA	Recommended Dietary Allowances
RE	retinol equivalents
sl	slice
sm	small
Tbsp	Tablespoon
TPN	total parenteral nutrition
tsp	teaspoon
USDA	United States Department of Agriculture